

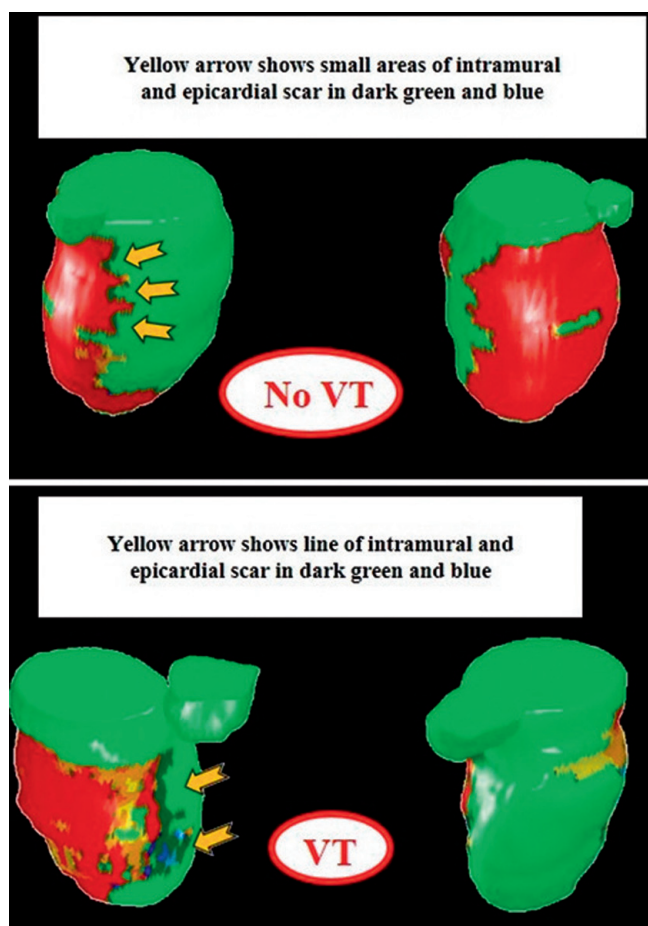
Introduction: The use of implantable cardioverter-defibrillators (ICD) is recommended to prevent sudden cardiac death (SCD) in patients with a reduced (≤ 30 -35%) left ventricular ejection fraction (LVEF) due to previous myocardial infarction (MI). Some patients, however, never receive adequate ICD intervention. We studied whether the characteristics of MI

scar, as assessed by magnetic resonance imaging (MRI), could predict the occurrence of ventricular tachycardia (VT).

Methods: Fifty-one patients (41 men, mean age=59±11 years) with a remote (>6 months) MI and a class I primary prevention indication for ICD implantation underwent a cardiac MRI study before implantation. Delayed contrast enhancement (DCE) was used to delineate post-MI scars. On the basis of manually outlined contours of left ventricular epicardial, endocardial and scar borders, the location and transmural extent of the scar were calculated.

Results: VT occurred in 15 patients (29%) after a follow-up of 43±24 months. There were no statistical differences between patients who experienced VT and those who did not for demographic data, LVEF, total myocardial and MI surface. At infarct borders, MRI showed areas with intramural and/or epicardial scar in all but one patients. Epicardial scar surface (3.6 ± 0.5 vs. 1.4 ± 0.3 cm²; $p=0.0005$) and intramural scar surface (4.0 ± 0.6 vs. 1.8 ± 0.4 cm²; $p=0.002$) were greater in patients with VT. In multivariate analysis, intramural and sub-epicardial scar surface remained significantly associated with the occurrence of VT (respectively: HR, 1.28/1cm²; CI, 1.10 to 1.51; $p=0.003$ and HR, 1.23/1cm²; CI, 1.01 to 1.51; $p=0.04$). Patients with intramural scar surface >1.65 cm² had lower 5 years VT free survival (33.8% vs. 100%; $p<0.0001$).

Conclusion: The presence of a critical surface of both intramural and epicardial scars at an infarct border may be key factors for the occurrence of VT after MI.



Abstract 0141 – Figure: 3D left ventricular reconstruction

0300

Relationship between spatial scar characteristics assessed by cardiac magnetic resonance imaging and cycle length of monomorphic ventricular tachycardia in post-infarct patients

Damien Voilliot (1), Freddy Odille (2), Damien Mandry (3), Marius Andronache (1), Olivier Huttin (1), Isabelle Magnin-Poull (1), Jean-Marc Sellaal (1), Arnaud Olivier (1), Vladimir Manenti (1), Beatrice Brembilla-Perrot (1), Hugues Blangy (1), Jacques Felblinger (2), Pierre-Yves Marie (3), Etienne Aliot (1), Nicolas Sadoul (1), Christian De Chillou (1)

(1) CHU Nancy Brabois ILCV, Cardiologie, Vandoeuvre Les Nancy, France – (2) CHU Nancy Brabois, IADI, INSERM U947, Vandoeuvre-Les-Nancy, France – (3) CHU Nancy Brabois, Médecine nucléaire, Vandoeuvre-Les-Nancy, France

Introduction: We have previously demonstrated that the spatial characteristics (intramural and epicardial components) of MI scar predicts the occurrence of monomorphic ventricular tachycardia (MVT) after a myocardial infarction (MI). We studied whether MI scar characteristics, as assessed by magnetic resonance imaging (MRI), was related to the minimum cycle length (mCL) of MVT.

Methods: We studied 50 patients (43 men, mean age 60±13 years) with previous MI, cardiac MRI study, primary (n=12) or secondary prevention indication of implantable cardiac defibrillator (ICD) and who experienced MVT. Delayed contrast enhancement (DCE) was used to delineate post-MI scars.

Results: MVT occurred 15±9 years after MI and the mCL was 303±49ms. MRI showed areas with intramural and/or epicardial scar (adjacent to areas showing endocardial or transmural scar) in all patient. Patients were classified depending on the median of the mCL (300ms). There were no statistical differences between the 2 groups for: gender, medication, indication of ICD, history of coronary artery revascularization, infarct location and for the following MRI parameters: left ventricular (LV) ejection fraction, LV end-diastolic volume, LV mass, total myocardial surface, total MI scar surface, transmural, endocardial or epicardial MI scar surface. In patients with a mCL>300ms, there was a trend for older age at first MVT episode (64 ± 2 vs. 57 ± 3 year; $p=0.06$) and a greater intramural scar surface (5.8 ± 3.7 vs. 2.9 ± 1.6 cm²; $p=0.002$). Age ($r=0.32$; $p=0.02$) and intramural scar surface ($r=0.47$; $p<0.001$) were associated with the mCL. After multiple linear regression, age and intramural scar surface remained significantly associated with the mCL (respectively: $\beta=1.3 \pm 0.6$; $p=0.03$ and $\beta=6.5 \pm 2$; $p=0.003$).

Conclusion: Our study suggests that the mCL of MVT may be related to age and to the intramural MI scar surface at the infarct border (figure next page).

0379

Premature ventricular beat-induced cardiomyopathy. Characteristics and prognosis factor for recovery after radio-frequency ablation

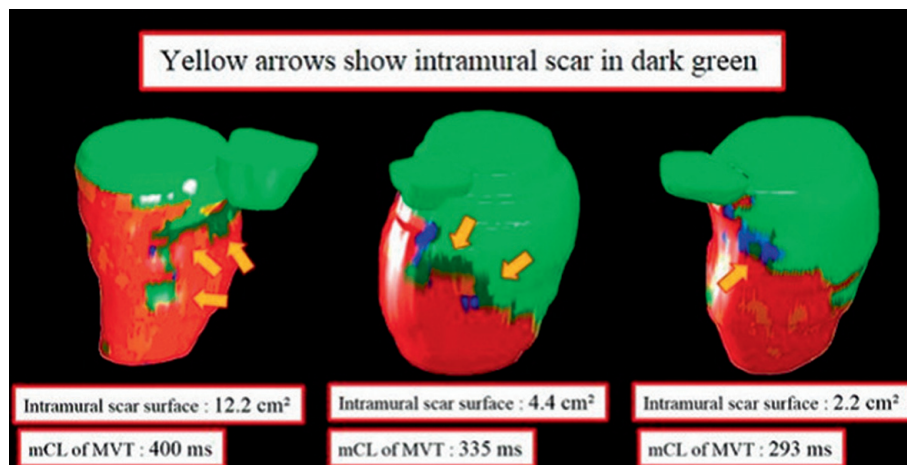
Marie Sadron (1), Philippe Maury (1), Frederic Sacher (2), Nicolas Lelouche (3), Patrizio Pascale (4), Alexandre Duparc (1), Pierre Mondoly (1), Anne Rollin (1), Nicolas Derval (2), Christelle Cardin (1), Pierre Jais (2), Meleze Hocini (2), Marc Delay (1), Michel Haissaguerre (2)

(1) CHU Toulouse Rangueil, Cardiologie, Toulouse, France – (2) CHU Bordeaux, Hôpital Haut-Lévêque, Cardiologie, Bordeaux, France – (3) CHU Henri Mondor-APHP, Cardiologie, Paris, France – (4) CHUV Lausanne, Cardiologie, Lausanne, Suisse

Introduction: Frequent premature ventricular beats (PVB) may induce cardiomyopathy (CM). Characteristics and prognosis factor for recovery after RF ablation remain debated.

Methods: 93 patients (74% men, 58±14 yo) with dilated CM associated with frequent isolated PVB were included. A group of 75 pts undergoing ablation for symptomatic PVB without significant cardiac disease serves as the control group.

Results: EF was 38±10% and left ventricular end diastolic diameter (LVEDD) was 63±8mm. One third have various associated cardiomyopathy. PVB burden was 27±12%. PVB arose from the left ventricle in 96 pts (LVOT 61, mitral 16, apex 7, septal 12) and from the right ventricle in 61 pts (RVOT 58) and multiple in 11. Epicardial ablation in the CS was needed in 25.



Abstract 0300 – Figure: 3D left ventricular reconstructions

In multivariate analysis, lack of palpitations (OR 9.09 [3.45-33.33]), VPB number > 20000 (OR 5.40 [1.98-14.70]), left ventricular origin (OR 4.12 [1.53-11.11]), epicardial location (OR 11.00 [1.92-62.50]), VPB right inferior axis (OR 2.31 [0.85-6.27]), baseline QRS width > 100ms (OR 3.66 [1.28-10.43]), VPB coupling interval > 500ms (OR 3.11 [1.14-8.55]) and polymorphic VPB (OR 10.40 [1.05-103.05]) were independently associated with CM compared to controls ($p < 0.05$).

Over a mean follow-up of 22 ± 20 months, 79% presented with a significant decrease of VPB (> 80% reduction). In these, EF increased (36 ± 9 to $51 \pm 12\%$, $p < 0.0001$) and LVEDD decreased (62 ± 7 to 56 ± 7 mm, $p < 0.0001$). Reversal of CM was defined by > 10% increase in EF. Only a VPB > 2mV (OR 19.2 [1.84-200.00], $p = 0.01$) was independently associated with reversal of CM in multivariate analysis.

Conclusion: Mechanisms leading to PVB-induced CM may involve lack of palpitations, a high VPB number, a left ventricular origin, an epicardial location, a VPB right inferior axis, a large baseline QRS duration, a long VPB coupling interval and polymorphic VPB. Reversal of CM after RF ablation may associate a high VPB amplitude and a shorter VPB coupling interval. This may help in selecting patients for RF ablation of suspected VPB-induced CM.

0395

Prevalence of early repolarization in congenital long QT syndrome. A combination of early and delayed repolarization

Mathieu Audoubert (1), Anne Rollin (1), Philippe Maury (1), Rémi Chauvel (2), Alexandre Duparc (1), Pierre Mondoly (1), Cristelle Cardin (1), Marc Delay (1), Marie Sadron (1), Frederic Sacher (2), Vincent Probst (3) (1) CHU Toulouse Rangueil, Cardiologie, Toulouse, France – (2) CHU Bordeaux, Hôpital Haut-Lévêque, Cardiologie, Bordeaux, France – (3) CHU Nantes, Cardiologie, Nantes, France

Introduction: early repolarization (ER) in Brugada or short QT syndrome is common and has been associated with a less favourable outcome. Even if apparently paradoxical, ER can also be seen in long QT (LQT) but prevalence and correlations to other variables are unknown.

Methods: ECG of 105 LQT patients (44 men, 36 ± 21 yo) and 269 age and gender matched controls (135 men, 36 ± 18 yo) were reviewed. LQT was diagnosed by a positive genetic testing ($n = 71$) or by showing abnormal T wave and long QT interval spontaneously or during epinephrin infusion in pts without discovered genetic mutation ($n = 34$). ER was defined by > 1mm J point elevation in the inferior or lateral leads with notch or slurring pattern.

Results: QT in lead II was 433 ± 68 msec in LQT patients and 338 ± 41 in controls ($p < 0.0001$) (QTc 446 ± 52 versus 377 ± 30 msec, $p < 0.0001$). Heart rate was lower in LQT patients (66 ± 14 vs 79 ± 19 bpm) ($p < 0.0001$). Twenty LQT

patients presented with resuscitated sudden death or torsades de pointes and 11 with syncope.

33/105 LQT patients (31%) had ER compared to 31/269 (12%) controls ($p < 0.0001$).

ER was more frequent in LQT men (19/44, 43%) compared to women (14/61, 23%) ($p = 0.03$) but was not correlated to age (41 ± 20 yo with ER vs 35 ± 21 bpm, $p = 0.17$).

LQT patients with ER had lower heart rates (61 ± 11 vs 69 ± 15 bpm, $p = 0.02$).

There was a trend toward longer QT in patients with ER (449 ± 73 versus 426 ± 64 msec in lead II, $p = 0.1$) but not for corrected QT intervals (442 ± 48 versus 448 ± 54 msec, $p = 0.5$).

There was more frequent ER in case of HeRG (14/26) than KCNQ1 (6/34) or KCNJ2 (2/10) mutations ($p = 0.008$).

ER in LQT patients was not correlated to symptoms or cardiac events (5/20 patients with SD, 3/11 in patients with syncope and 25/72 asymptomatic LQT) ($p = 0.6$).

Conclusion: ER is very common in LQT patients and is related to the gender and to the heart rate but not to age or to the corrected QT duration. ER is not correlated to cardiac events in this series but may be to HeRG mutations

0555

Arrhythmic risk stratification and prognostic value of programmed ventricular stimulation in arrhythmogenic right ventricular cardiomyopathy/dysplasia

Carole Maupain, Nicolas Badenco, Guillaume Duthoit, Xavier Waintraub, Thomas Chastre, Caroline Himbert, Françoise Hidden-Lucet, Estelle Gandjbakhch (CHU La Pitié-Salpêtrière-APHP, Institut de Cardiologie, Paris, France)

Background: The role of programmed ventricular stimulation (PVS) in arrhythmic risk stratification is unclear in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D).

Objective: To determine clinical factors associated with inducibility of PVS and determine its pronostic value in the overall population and in three risk groups.

Methods: Between 2000 and 2010, 150 consecutive patients systematically benefited PVS at diagnosis. Predictors for PVS inducibility were studied. Risk factors for arrhythmic events were then determined by Cox regression in the entire population and in three risk groups.

Results: VT inducibility was significantly higher for males ($p = 0.007$), symptomatic patients ($p < 0.001$) especially those with syncope ($p = 0.004$), patients who had spontaneous ventricular tachycardia (VT) ($p < 0.001$) and right ($p < 0.001$) or left ($p = 0.03$) ventricular dysfunction.